

# Methods for Reducing Lead Exposure in Young Children and Other Risk Groups: An Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning

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As part of a Congressionally mandated report on U.S. childhood lead poisoning prepared by the Federal government (U.S. Agency for Toxic Substances and Disease Registry [ATSDR]), the authors have analyzed the relative effectiveness of measures to reduce source-specific lead exposure of U.S. children. An integrated overview of this analysis is presented in this article. Two national actions, the Federally mandated phasedown of lead in gasoline by the U.S. Environmental Protection Agency and the voluntary phasedown of lead use in domestic food can production, are examples of centrally directed initiatives that have been relatively successful in limiting childhood lead exposure in the U.S. Efforts to abate lead-based paint exposure of children have largely failed. This is especially true for the nation's 21 million residential units with the highest lead content paint. Similarly, abatement of lead exposure from contaminated dusts and soils has generally been unsuccessful. Comprehensive measures to reduce lead exposure from drinking water in residences and public facilities, e.g., elementary schools, are only now being promulgated or implemented. The full extent of their effectiveness remains to be demonstrated. There are many miscellaneous but potentially severe exposure sources that are difficult to control but require attention, such as poorly glazed foodware and ethno-specific preparations.

## Introduction

In mandating a report to Congress (1) on U.S. childhood lead poisoning, Section 118(f) of the 1986 Superfund Amendments and Reauthorization Act (SARA) directed the Federal government's Agency for Toxic Substances and Disease Registry (ATSDR) to examine methods and alternatives for reducing environmental lead exposure in young children in the U.S. This paper concerns Chapter IX of ATSDR's report to Congress (2), with some further updating. This topic encompasses many environmental and social issues, and only a limited number of them could be discussed in the report to Congress.

One clear message from a number of chapters in the report to Congress (1) is that significant childhood lead exposure persists for a number of lead sources and pathways. For other sources, specific measures with major conse-

quences for exposure control have been put in place in the U.S. These measures are helping to reduce some of the original levels of exposure and toxicity in identifiable segments of populations at special risk.

Lead exposure abatement can be examined with regard to various levels of effectiveness. Is it simply the lowering of population blood lead (PbB) levels below some value associated with an adverse health risk? Alternatively, should reducing population exposure also provide some margin of safety? This safety margin is desirable for obvious reasons, not the least of which is the strikingly small size of this margin in many individuals, i.e., between PbB levels in many subjects and levels at which health effects are seen. Future information may well cause further downward revisions in acceptable levels of PbB, providing a second argument for an adequate current safety margin.

Exposure prevention is described here as either primary or secondary preventive measures. Tertiary components of exposure prevention, as defined by others, are presented in this article as a part of secondary methods. Specific components of each type of exposure prevention method are depicted in Table 1.

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**Table 1. Categorical tabulation of components of primary and secondary prevention of lead exposure in children and related U.S. risk groups.**

Type of prevention method	Components of method
Primary	
Environmental	Lead in paint Lead in ambient air Leaded gasoline combustion Point source emissions Lead in dusts/soils Lead in drinking water Lead in foods
Environmental/biological	Source controls augmented by community-nutrition interventions, i.e., nutritional supplementations, for calcium and iron
Secondary	
Environmental	Case finding Screening programs Environmental follow-up Event-specific exposure abatement
Environmental/biological	Nutritional assessment and follow-up on ad hoc identification basis
Extra-environmental	Legal actions and strictures

In the case of lead exposure, primary prevention involves both preventing entry of the lead source and its removal, reduction, or avoidance of contact once present. By contrast, secondary methods of prevention are basically reactive in nature, i.e., they comprise a cluster of responses to existing and identified problems.

Primary and secondary prevention strategies are further differentiated as to either environmental exposure exclusively or environmental control in tandem with biological approaches. The latter involve reduction of *in vivo* exposure and toxicity risk. In lead exposure, for example, nutritional factors such as adequate iron, calcium, and phosphorus can reduce, to some degree, lead absorption from the gastrointestinal tract in children. Optimizing child nutrition, however, is no substitute for environmental exposure abatement.

Examples of primary and secondary prevention of lead exposure are found in the case of leaded paint. The banning of toxic levels of lead in newly manufactured paint, in 1977, by the U.S. Consumer Product Safety Commission illustrated primary prevention of lead exposure. This action, while a primary prevention initiative, did nothing for the enormous reservoir of leaded paint in tens of millions of U.S. dwellings. This exposure threat therefore requires combined primary and secondary approaches. Primary steps include leaded paint abatement and preventing the flaking of old paint. Screening for actual leaded paint exposure, by contrast, is considered secondary prevention, as are steps to minimize child contact with existing leaded paint.

Past and current problems with lead as a U.S. public health problem are traceable to failures in primary prevention mechanisms. For example, adequate safety assessments for leaded paints and leaded gasoline, as now commonly defined, were not originally applied to these sources. According to Rosner and Markowitz (3) and

Hamilton et al. (4), who examined the use of leaded gasoline at either end of a 60-year span, the introduction of lead alkyls as a gasoline antiknock additive was permitted in the absence of any credible public health risk assessment. Present U.S. regulatory practices would not permit very many uses of lead if it were in new products.

## Primary Prevention Measures for Lead Exposure

This section deals with the environmental control of lead in paint, lead in the atmosphere from leaded gasoline and stationary sources, dusts and soils, water and food. It also includes use of both environmental and nutritional measures.

### Primary Prevention Using Environmental Measures

Primary prevention as applied to lead exposure to various sources has actually been a hybrid of conventional primary prevention measures and *post hoc* efforts that resemble secondary prevention approaches.

**Lead in Paint.** National and other regulatory actions to control leaded paint exposure were only instituted after childhood lead poisoning cases associated with leaded paint ingestion had been recognized (5). Control actions are divided into Federal and non-Federal controls.

**Federal Actions in Preventing Paint Lead Exposure in Young Children.** Federal action for primary prevention of childhood leaded paint exposure are mainly those of the U.S. Department of Housing and Urban Development (HUD) and the U.S. Consumer Product Safety Commission (CPSC). These actions were mandated by Congress through diverse legislation.

The principal role of CPSC in lead control was to mandate reduction of lead levels in paint to 0.06% in 1977. This measure only affected the rate of new input of leaded paint into U.S. housing and public building stock, since CPSC's mandate does not address the preexisting paint lead burden in U.S. housing stock, nor does it cover paints not sold in interstate commerce. Reduction to a level of 0.06% followed an unofficial voluntary restriction by the manufacturers themselves to a 1% lead content in the late 1950s. However, between the 1950s and 1977, paint stocks with lead in excess of this level continued to be produced (5). This level of 1% (as dry solid) still amounted to 10,000 ppm lead, a level sufficient to produce elevated risk of systemic exposure (2).

A lingering problem with leaded paint is the disposition of old retail stock that has high lead content. CPSC, for example, is not authorized to act against salvage, close-out, and bankruptcy sales if stock was manufactured before the June 22, 1977, effective date of the 0.06% standard. Therefore, some high lead paints may still be in retail channels.

The Connecticut Department of Consumer Protection, for example, has noted that lead-based paint can reach the

market in higher amounts than expected (Communication of Department of Consumer Protection, State of Connecticut, to Jane S. Lin-Fu, HHS, September 17, 1985). Connecticut found lead-based paint that was over 22 years old on retail shelves during 1985.

Discount and salvage outlets will have bought close-out inventories and kept lead-based paint in the consumer pipeline to some extent. Of concern also is the fact that paint producers are permitted to market a "sludge" paint from new materials plus residues from vats. If these residues are from lead-containing industrial products, then the ultimate lead level in the sludge paint may exceed the CPSC limit of 0.06%.

In contrast to CPSC's role, that of HUD is directed to leaded paint already present in either public housing or housing involving Federal assistance. However, HUD did restrict the use of high lead levels in paints in housing stock under its jurisdiction. The 1971 Lead-Based Paint Poisoning Prevention Act (LPPPA) (6) authorized HUD action to prohibit the use of leaded paint in Federal or Federally assisted construction or rehabilitation; relevant HUD regulations were adopted in 1972. A major statutory step forward in HUD's responsibilities was mandated in Section 302 of the Act (6), added in 1973, which required HUD to set up procedures for leaded paint abatement in existing housing stock. Here also, jurisdiction was limited to Federally connected housing.

In 1973 and again in 1976, HUD acted in two ways under provisions of Section 302: warnings to purchasers and tenants of HUD-associated housing as to "immediate hazard" in housing built before 1950, and prohibiting lead-based paint at a level above 0.5% (prior to the 0.06% level as of June 22, 1977). Recently, HUD has become more involved as a result of the outcome of 1983 Federal court action (7). This action successfully challenged HUD regulations so as to include essentially all lead-painted surfaces as an "immediate hazard" rather than just those conditions associated with deteriorating painted surfaces.

HUD has promulgated three rules that extend its activities in this area: *a*) lead paint hazard elimination in public and Indian housing (8); *b*) lead paint hazard elimination in FHA single- and multifamily units and Section 8 housing/housing voucher and rehabilitation, FHA single- and multifamily property disposition (foreclosure) programs (9), and *c*) lead paint hazard elimination in various community-based Federal grant and related programs (10).

Collectively, these new actions address virtually the full spectrum of U.S. housing activity in which HUD has some assistance role. However, no Federal action exists to reach directly into fully private sector housing except for control of the lead level in new paint offered for sale (see above).

The new regulation concerning public and Indian housing includes required inspections for defective paint surfaces in units with children less than 7 years old and require inspections for chewable and defective surfaces if a child has an elevated PbB level. The test threshold for paint lead in all cases is 1 mg/cm<sup>2</sup> lead. These regulations also require accurate use of lead detectors by competent operators. Hazard abatement, i.e., leaded paint removal, is required when a child is identified with an elevated PbB in the dwell-

ing, in common areas, or in public child care facilities within control of public housing.

HUD activity in FHA-supported and similar housing in these regulations had a 1973 construction cutoff, i.e., housing built in this year and earlier is covered under the action, but this date was subsequently changed to 1978. Inspection for defective surfaces, as with the public/Indian housing action, does not require X-ray fluorescence analysis, but the chewable, protruding surfaces do. Further details can be found in the Federal Register notice (9). Testing and abatement actions for FHA-assisted housing are triggered by change in ownership and continuation of Federal mortgage insurance. If a leaded paint-contaminated unit remains in its present ownership or is bought through non-Federal financing, then these requirements do not apply.

The third action requires that Community Development Block Grant, Urban Development Action Grant, Secretary's Fund, Section 312 Rehabilitation Loan, Rental Rehabilitation, and Urban Homesteading Program applicants must carry out lead paint analysis and abatement steps in order to receive funds within the programs. Both this cluster of HUD community grant programs and those involving FHA-related assistance place abatement costs primarily on the private sectors involved in the housing transactions.

Most recently, HUD has taken further steps toward paint hazard elimination (11) as directed by Section 566 of the Housing and Community Development Act of 1987 (12). This rulemaking amends a number of current regulations, changes definition of abatable surfaces to include exterior as well as interior surfaces, and advances the construction cutoff date to 1978 from 1973 (see above). Finally, and as required by PL 100-242 (12), HUD must report to Congress by December 1989 on safe and effective abatement methods and a comprehensive inspecting and paint lead removal plan.

While these recent actions suggest a more concerted effort to attack the leaded paint hazard, quantification of the likely or estimated impact of the three rulemaking actions is important. Table 2 provides estimates of the number of units and associated abatement costs in public housing, at a paint lead removal action level of 1 mg/cm (13). About 308,000 units are estimated to require abatement across all unit age categories with an aggregate cost of \$380.1 million in 1986 dollars. This figure appears to be too low.

Table 3 presents the estimated number of units requiring lead abatement for each year, 1987 to 1991, and the projected cost in these years for FHA single-family units (14). For all housing ages, 171,300 units are estimated to require abatement for each of the 5 years, and total 856,500 units with a cost of about \$2 billion. Single-family, FHA-insured units are but one category in this particular HUD action. Miller and Toulmin (15) have estimated that for 1987 to 1991, all of these FHA categories will involve an outlay of \$2.57 billion. Of these amounts, about 95% will have to be paid by buyers and/or sellers in the private sector.

**Municipal and State Actions in Leaded Paint Exposure.** In 1951, the city of Baltimore prohibited leaded paint use on interiors of dwelling units and, in 1958, required war-

**Table 2. Abatement costs and number of units for different site categories at a leaded paint threshold of 1.0 mg/cm<sup>2</sup> in public housing.<sup>a</sup>**

Construction year	Family dwelling units		Cabinet surfaces		Housing project units		Common activity sites	
	Number	Cost, \$ millions	Number	Cost, \$ millions	Number	Cost, \$ millions	Number	Cost, \$ millions
Pre-1950	81,379	86.7	5,399	3.4	11,239	25.7	413	0.7
1950-1959	111,688	108.6	3,609	2.2	16,808	61.0	425	0.5
1960-1972	114,587	62.8	0	0	11,361	28.2	457	0.3
Total	307,654	258.1	9,008	5.6	39,408	114.9	1,295	1.5

<sup>a</sup>Adapted from Wallace (13). Number of units indicated is 48.9% of the total of 629,004, and total cost is \$380.1 million.

**Table 3. Estimated abatement costs and number of units for different site categories at a lead paint threshold of 1.0 mg/cm<sup>2</sup> in single-family FHA housing units.<sup>a</sup>**

Year built	Number of units				
	Year of abatement				
	1987	1988	1989	1990	1991
1960-1972	20,500	20,500	20,500	20,500	20,500
1950-1959	55,900	55,900	55,900	55,900	55,900
Pre-1950	94,900	94,900	94,900	94,900	94,900
Total number	171,300	171,300	171,300	171,300	171,300
Total cost, \$ thousands	388,400	388,400	388,400	388,400	388,400
Cumulative cost, \$ thousands	388,400	776,800	1,165,200	1,553,600	1,942,000

<sup>a</sup>From Miller and Toulmin (14).

ning labels on cans of leaded paint already in the market pipeline (15). By that time, the paint industry had introduced titanium dioxide as a substitute pigment for basic lead carbonate in paint, but lead-based paint continued to be made into the 1970s.

Retroactive regulation at any level of jurisdiction, i.e., states or cities, for paint lead already in U.S. housing stock has been infrequent and variably enforced. In the early 1970s, Philadelphia, PA, had a primary prevention ordinance directed at removing leaded paint up to 5 feet above the floor in any unit with leaded paint. However, the city eventually discarded such prophylactic removal in favor of abatement only after demonstrated toxicity in children who lived in the units.

Among the states, a number have attempted to attack the problem by various lead paint poisoning control statutes. These have generally produced mixed results. For example, Massachusetts first banned lead in any unit in which children younger than 6 years of age were living, but various problems with landlord cooperation and limited funding for enforcement resulted in control limited to secondary prevention; that is, intervention only after demonstrated instances of toxicity (16). Subsequently, in late 1987, Massachusetts enacted stronger laws to strengthen the lead paint hazard identification and protocols for lead paint abatement. It is too early to judge their effectiveness.

In many states, there are large inventories of old housing requiring leaded paint removal. Summary statistics provided by the Commonwealth of Massachusetts to ATSDR, shown in Table 4, permit some observations as to the magnitude of the problem and the rate of its remediation. Of interest is the activity level of lead removal programs compared with

the number of pre-1940 housing units, that is, lead-painted units with high lead content. The table indicates that the selected cities of the Commonwealth have a total of 450,339 pre-1940 units (those with the highest lead content). Over the period January 1982 to June 1986, only 2260, or 0.5%, of these units were subjected to lead abatement. We are not aware of the level, if any, of lead removal carried out under Massachusetts statutory provisions but occurring outside the reported programs.

Statutes such as that of Massachusetts can be employed in concerted action by specific community groups. For example, a tract of high-risk, lead-painted housing in the Jamaica Plain area of Boston was systematically examined in 1981, the children involved were screened for lead toxicity, and then 50% of the suspect housing was treated to remove lead. The joint efforts of the Harvard School of Public Health, which did the community assessment, and the Legal Aid Society, which used the Massachusetts statutory sanctions, forced landlords to comply (17).

**Lead in Ambient Air: Leaded Gasoline Combustion and Point Source Emissions.** EPA has had regulatory authority over the use of lead in gasoline since 1973 (18). In 1975, EPA classified lead as a criteria pollutant, a designation reserved for pollutants whose public impact is such that control is required by ambient standards rather than by site-specific emission controls. Several parallel actions were

**Table 4. Summary of total pre-1940 lead-painted housing versus deleading activity in selected Massachusetts communities for 1982-June 30, 1986.<sup>a</sup>**

City	Pre-1940 units <sup>b</sup>	Units deleaded in					Total
		1982	1983	1984	1985	1986 <sup>c</sup>	
Boston	179,391	221	175	136	201	152	885
Worcester	43,555	148	100	99	142	67	556
Springfield	36,239	40	41	29	34	2	146
New Bedford	29,536	9	21	16	10	1	57
Fall River	28,502	6	5	2	1	0	14
Somerville	26,806	9	7	1	4	9	30
Lynn	26,006	20	29	21	35	12	117
Lowell	23,356	—	—	—	—	—	153 <sup>d</sup>
Lawrence	19,916	—	—	—	—	—	300 <sup>d</sup>
Newton	18,516	0	1	1	0	0	2
Total	450,339	453	379	305	427	243	2260

<sup>a</sup>Summary statistics: childhood lead poisoning prevention program. Commonwealth of Massachusetts, as provided by Cosgrove to ATSDR, December 10, 1986 (2). Communities ranked by number of pre-1940 units

<sup>b</sup>All pre-1940 units are assumed to have leaded paint at significant levels.

<sup>c</sup>To June 30, 1986.

<sup>d</sup>Total only supplied.

being pursued in 1975 under the aegis of either Section 108 or 109 of the Clean Air Act, as amended (19), which authorized the EPA Administrator to set ambient air standards for lead.

In addition, Section 211(c)(1) of the Act (19) authorizes the Administrator to "control or prohibit the manufacture . . . or sale of any fuel additive" if its emission products cause or contribute to "air pollution which may be reasonably anticipated to endanger the public health or welfare" or "will impair to a significant degree the performance of any emission control device or system. . . in general use."

Since the mid-1970s, the use of lead in gasoline declined mainly as a result of the increase of lead-sensitive, emission control-equipped vehicles in the U.S. domestic fleet. National data are best illustrated by results of the Second National Health and Nutrition Examination Survey (NHANES II) (20) for the general population and children at high risk assessed in U.S. Centers for Disease Control screening programs (21). The NHANES II data indicated a generalized, cross-population decline in PbB levels of 37%, an average drop of about 5.4  $\mu\text{g}/\text{dL}$  (22). In 1978, an ambient air lead standard of 1.5  $\mu\text{g}/\text{m}^3$  was promulgated—a considerable drop from the earlier standard. This standard also provided a means for controlling point-source emissions from smelters and similar operations.

In 1982, EPA set forth new rules (23) that, among other things, reduced the lead content of gasoline to 1.1 per liquid gallon. In further action, effective January 1, 1986, EPA revised the phasedown of lead in gasoline to 0.1 g per liquid gallon. The decline in gasoline lead from these actions is expected to impact the number of children whose PbB levels fall below certain toxicity risk ceilings, including the 1985 CDC action level of 25  $\mu\text{g}/\text{dL}$ . Prevalence modeling estimates by EPA (24) project sizable declines in the numbers of children with PbB levels above 15, 20, and 25  $\mu\text{g}/\text{dL}$ , owing to gasoline lead phasedown.

EPA is also examining its 1978 lead standard of 1.5  $\mu\text{g}/\text{m}^3$  in ambient air. Any downward revisions will reduce atmospheric inputs, mainly near stationary sources. Controls on lead input from mobile and stationary emissions control new inputs. Populations will continue to be at risk for exposure from lead-contaminated dust and soil, arising from past air lead fallout (as well as from leaded paint weathering and chalking).

**Lead in Dusts and Soils.** The principal prevention measures for lead-contaminated dust and soil exposures have been directed at the generators of lead in paint, leaded gasoline, and stationary source emitters. Currently, only very limited regulatory action has been specifically directed at controlling lead in dust and soil. In the case of lead-containing waste sites, Chapter X of the report to Congress (2) describes Superfund activity and Appendix F of the report lists sites that are due for cleanup and which also contain lead in soil. Several factors have contributed to this lack of regulation. First, dust and soil have not been traditionally recognized in public health actions or policy as specific, potentially major sources or pathways of childhood lead exposure. These sources are complex and still require more precise quantitative characterization. Second, legal

and other societal sanctions that are not enforced allow primary contributors such as leaded paint to continue to contaminate residential dusts and soils. One impediment to regulatory or legal control of lead in dusts and soils has been the relative paucity of studies showing how specific primary contributors quantitatively affect given dust and soil contamination levels. Duggan and Inskip (25) have reviewed dusts and soils versus childhood exposure in detail.

Recent reports document that lead levels in these media are quantitatively related to PbB levels (22). Charney et al. (26) have shown that PbB levels can be reduced through indoor dust abatement but only to a certain point. Milar and Mushak (27) have shown a relationship between workplace dust inadvertently brought home by lead battery plant workers and PbB levels in their young children. Similarly, the study by Ryu et al. (28) shows household contamination via secondary transport from the workplace and lead transfer to infants. Reports of the Cincinnati, OH, prospective lead studies, concerned with childhood lead poisoning in that city, have shed considerable light on relationships among pathways for household dust, lead on the hands of children, and socioeconomic factors concerning leaded paint as the likely primary contributors (29–32). Clark et al. (31) have shown that dust lead is best correlated with lead on the hands of children, and their results point to dust lead abatement as a key factor in reducing lead hazards in housing.

The focus of most studies to date has been lead abatement methods that are applicable to individual lead paint-containing units. Field studies are therefore needed to provide evidence that macro rather than micro control strategies are effective means of lead abatement in areas larger than a single home or several homes. Mobility of lead in dust and soil prevents a straightforward application of methods for single unit abatement to a neighborhood or even larger areas. Additional field surveys may also be needed to define blood lead-source lead relationships. Past attempts to define soil and dust lead in terms of precise proportional contributions of paint lead and airborne lead, when both input sources were present, have been unsuccessful for various reasons.

The 1986 SARA provides for the funding and execution of demonstration projects to address the problem of area-wide soil (and dust) lead in urban tracts. In response, the U.S. EPA has provided for a small group of major demonstration projects in several large U.S. cities, but results are still very preliminary.

**Lead in Drinking Water.** EPA is required by the 1974 Safe Drinking Water Act (SDWA) (33) to set drinking water standards with two levels of protection, labeled primary and secondary standards. The primary standards for drinking water, related to human health, define contaminant levels in terms of maximum contaminant level (MCL) or treatment requirements. MCLs are limits enforceable by law and are to be set as close as possible to maximum contaminant level goals (MCLGs). MCLGs are levels essentially determined by relevant toxicologic and biomedical considerations independent of how feasible attaining the levels may be.

In the 1986 amendments to the 1974 SDWA, EPA was directed to tighten the drinking water standards for various

substances, including lead. The current MCL for lead is 50  $\mu\text{g/L}$  of water (34). In response, EPA has proposed a revision of the MCL from 50 to 5  $\mu\text{g/L}$  water, measured at the water system's outlet rather than the residential tap (35). The latter is principally involved through the mechanism of an action level, i.e.,  $>10 \mu\text{g/L}$ . This is determined by a two-tier tap water sampling in a community, depending on population. If a community's sampling average exceeds 10  $\mu\text{g/L}$ , then corrosion control is required. Central corrosion control is an especially important part of the proposed changes. In addition, EPA is also proposing to consider the removal of lead service connections and goose-necks (connections from the street line to house lines). Other options may be considered and selected, and it is not possible at this time to predict the final form of tap water lead rule changes.

In addition to the pending rule on drinking water lead *per se*, the 1986 SDWA (33) amendments banned the use of lead solder and other lead-containing material in household plumbing when residences are connected to public water supplies. States must enforce the ban or be subject to a loss of Federal grant funds.

In late 1988, the Lead Contamination Control Act of 1988 was enacted (36), which contained both primary and secondary lead exposure prevention provisions. These include the recall of lead-containing public water coolers, the screening of school tapwater lead levels, and assistance to states for testing and abating lead in school drinking water.

EPA's Office of Policy Analysis (37,38) has carried out a detailed assessment of lead in drinking water from public water supplies. As noted in the report to Congress (1), about 20% of the population has tap water lead levels above a level of 20  $\mu\text{g/L}$ .

Corrosive drinking water is quite common to densely populated U.S. urban areas, and the U.S. EPA (37) has estimated that about 62 million Americans consume such water. A useful U.S. case study for primary prevention of exposure to lead in drinking water at the community level is that of the Boston water system. In the 1970s, the Boston water authority began efforts to reduce corrosivity, since many of the occupied housing units in the city had lead plumbing, and the water supply was highly corrosive. These efforts considerably reduced the amount of lead in tap water (22,37). The U.S. EPA (37) has estimated that the treatment to reduce corrosivity costs just 25% of the value of the health benefits realized from reduced lead exposure, that is, a benefit-to-cost ratio of 4:1.

While the Federal actions described above are expected to have an impact on potential childhood lead exposure, the final form of the proposed changes and their effective implementation will determine the efficacy of lead control in U.S. water supplies.

**Lead in Food.** Some quantity of lead in food and beverages is ingested by virtually the entire U.S. child population. As noted in the report to Congress (1), about 5% of these children were estimated to have a lead intake high enough to potentially increase PbB levels to those associated with early health impacts. Consequently, prevention measures that limit lead exposure from food are important.

Regulating lead contamination in foods has been the responsibility of the U.S. Food and Drug Administration (FDA) for several decades. Such control began with the identification of lead-containing pesticide residues on sprayed fruits. Collectively, FDA actions from the 1970s onward have targeted either control through setting total lead intake goals or efforts directed at known significant sources of lead inputs into foods.

In 1979, FDA set a long-term goal of less than 100  $\mu\text{g/day}$  for reducing the daily lead intake from all foods for children 1 to 5 years old (39). This is a maximum permissible intake for any child and not a mean intake for all children. To achieve this goal within the shortest feasible time, attention has been focused on *a*) establishing permissible lead residues in evaporated milk and evaporated skim milk; *b*) setting maximum levels for lead in canned infant formulas, canned infant fruits and vegetable juices, and glass-packed infant foods; and *c*) establishing action levels for other foods. Along with these activities, FDA attempts to monitor and enforce controls on food-related materials. This includes leaching from pottery glazes and food utensils.

Lead can enter the food supply during production, processing, or distribution. The U.S. EPA (22) has determined that during these activities, the lead content in food may be increased 2- to 12-fold over background levels. Processing is the major pathway for contamination, especially lead leached from lead-soldered cans. Since World War II, the ratio of lead to tin in this soldering material has remained at 98:2.

FDA activities, to a large extent, consist of establishing voluntary cooperation from domestic food manufacturers and processors, and much of the data is provided by the industry. Clearly, lead in food due to leaching from lead-soldered sources has been significantly reduced (Table 5).

The percentage of food cans that are lead-soldered continues to decline. Table 5 shows the percentages from 1979 through the first quarter of 1986 and also for the end of 1988. The percentage was very high in 1979—over 90%—but had declined by the end of 1988 to approximately 6%. Recent data provided to FDA by the National Food Processors Association (NFPA) (40) indicate about a 77% reduction in lead from canned food during the period 1980 to 1985. Table 5 does not include imported canned foods;

Table 5. Percentage of lead-soldered cans in all U.S. manufactured food cans from 1979 to 1985<sup>a</sup> and 1988.<sup>b</sup>

Year	Total food cans, millions	Lead-soldered cans, millions	Percent of total
1979	30,543	27,576	90.29
1980	28,432	24,405	85.84
1981	27,638	20,516	74.23
1982	27,544	17,412	63.21
1983	26,942	13,891	51.56
1984	28,121	11,683	41.55
1985	27,767	8,769	31.58
1986 <sup>c</sup>	6,517	1,807	27.72
1988 <sup>b</sup>	28,071	1,626	5.79

<sup>a</sup>From Can Manufacturers Institute data to U.S. FDA (2).

<sup>b</sup>From Can Manufacturers Institute (personal communication, April 1989).

<sup>c</sup>First quarter, 1986.

**Table 6. Age- and sex-dependent diet lead intakes in the U.S. at two time periods.<sup>a</sup>**

Age, body weight	Sex	Lead intake, $\mu\text{g/kg/day}$		Change (%) $\mu\text{g/kg/day}$
		1982-1984	1984-1986	
6-11 months, 9 kg		1.70	1.11	-0.59 (35)
2 years, 13 kg		1.60	1.00	-0.60 (37)
14-16 years, 54 kg	F	0.48	0.30	-0.18 (33)
14-16 years, 60 kg	M	0.63	0.38	-0.25 (40)
25-30 years, 60 kg	F	0.43	0.27	-0.16 (39)
25-30 years, 76 kg	M	0.48	0.29	-0.19 (40)
60-65 years, 64 kg	F	0.42	0.25	-0.17 (40)
60-65 years, 76 kg	M	0.44	0.26	-0.18 (42)

<sup>a</sup>From FDA Division of Toxicology, Communication of Internal Tabulations to ATSDR, April 23, 1987, based on Total Diet Study results (2). Revised Total Diet Study points, eight collections.

there are no data for this contribution to lead in food.

Some changes in steps causing the lead contribution from the food processing industry were not taken until after 1981/1982. In the period 1980 to 1985, lead in canned food was reduced 77% (40), and lead in infant foods was reduced considerably (41). Recent data provided by FDA update the age-dependent reduction found in data from the Total Diet Study between 1982 to 1984 and 1984 to 1986. Table 6 gives total diet lead changes with the percentage decline for various age-sex categories.

Currently, the FDA surveys lead contamination in the U.S. food supply by means of the Total Diet Study, in which multiple food categories are included. Data for the ongoing Total Diet Study are based on samples that are still very small in relation to the enormous quantities of food units produced and consumed in the United States. They may not account adequately for variation by region and multiplicity of processors. The types of food items selected for testing also may not reflect the variations in food selection and consumption patterns among various segments of the U.S. population. The level of lead in food may consequently be smaller or greater than indicated.

Lead contamination of liquids and foods by use of poorly fired lead-glazed pottery can occur, but it is not possible to quantitate the effectiveness of any FDA controls concerning these objects. Similarly, use of lead-based folk medicine preparations by various ethnic groups are quite difficult to control.

### Primary Prevention of Exposure Using Combined Environmental and Biological Measures

Biological factors can suppress lead uptake into the body or enhance its excretion. When these factors are nutrients that have well-established interactive relationships with lead uptake and toxicity, such nutrients can be used to reduce internal or *in vivo* exposure. Such factors, when employed in a prophylactic, communitywide way, can also be viewed as an example of primary prevention. When these factors are exploited on an *ad hoc* basis in children or families where lead poisoning has occurred, their use becomes more a secondary prevention measure.

As discussed by U.S. EPA (22) and Mahaffey et al. (42), a number of nutritional factors suppress lead absorption and toxicity in test animal and human populations. However, only the nutrients iron and calcium can realistically be considered in the context of preventive community medicine for high-risk populations.

Results of numerous studies have shown that both calcium and iron nutritional status in young children is inversely related to the lead absorption level. Most of these studies are discussed in the EPA document (22). A more recent analysis of the NHANES II survey data showed a significant negative correlation between calcium status and PbB levels in a group of children under 11 years of age (42). As Mahaffey (43) has indicated, improving the nutritional status of children with high risk of exposure/toxicity does increase the effectiveness of environmental lead abatement. On the other hand, maintenance of optimal nutrition would mainly shift the lead level required for toxicity, i.e., the dose-effect curve, and would not eliminate lead toxicity risk.

Other nutrients that offset lead toxicity may not be particularly useful or advisable in this connection. Levels of phosphorus in most diets seem high enough to suggest intake is at adequate levels in poorer children, which is borne out by the Mahaffey et al. (42) examination of the NHANES II data for children. Vitamin D enhances lead uptake in the gut, but its intake is essential to health and cannot be reduced.

At present, no formal nutritional programs specifically geared to minimizing lead uptake or toxicity have been implemented in the United States. Nutrition monitoring and maintenance to minimize lead exposure are probably best done in a program of overall nutritional care, for example, the Women, Infants, and Children (WIC) nutrition program. The level of public funding and other support for such programs determines their potential in reducing net lead exposure. The argument can also be reversed to show that increased nutritional impairment for those at high risk for lead poisoning will enhance exposure and toxicity risk in that population. It is well known that nutrient deficiencies enhance lead uptake and toxicity (22,42).

## Secondary Prevention Measures for Lead Exposure

### Environmental Lead Control

This section principally addresses lead screening programs and other components of early intervention in exposure and toxicity, particularly environmental lead source identification and lead hazard abatement.

**Screening Programs and Case Finding.** The 1971 LPPPA, as noted by Farfel (5), did not specifically dictate health-based (secondary prevention) versus hazard-abatement (primary prevention) steps to be taken to ameliorate lead poisoning in U.S. children. While Title II of the Act (6) authorized grants to the responsible agency to remove leaded paint on a tract basis in high-risk neighborhoods, no funding for this purpose was provided. The Department of Health, Education, and Welfare emphasized intervention,

including medical management if necessary, for documented toxicity.

Various lead screening programs in high-risk areas of the United States, their history, and their quantitative aspects were discussed in the report to Congress (1). The focus here is on their role as secondary prevention instruments. During the time when screening programs were administered by the U.S. CDC, i.e., until fiscal year 1982 when CDC control ended, about 4 million children were screened nationwide, and about 250,000 children were registered as having met toxicity risk criteria then in use. The screening program was estimated to have surveyed about 30% of the high-risk children. Detection rates for positive toxicity through screening cases are considerably below those found in the NHANES II survey. Reasons for this difference are noted in the report to Congress (1).

Screening and early detection of exposure and toxicity have clearly reduced the rates of severe lead poisoning (21). However, chronic lead exposure and lower-level lead toxicity appear more resistant to such secondary prevention approaches. The persistence of these problems is predictable, given the levels and types of unabated exposure remaining in the United States (1).

In 1981, Federal resources for screening were put under the program of the Maternal and Child Health Block Grants to States. Although the amount of Federal funds for lead screening in such grant programs to states was estimated by one source to have been reduced by 25% (5), a precise figure cannot be readily given. Allocations of funds for particular projects within a block grant are determined by the States, and data are not systematically collected on these State decisions.

Information on any adverse impact of this initial reduction in Federal resources on screening effectiveness appears to be inconclusive. Although the total number of screening programs in the nation has decreased from the time of CDC administration (2), in some States and localities the number of children being screened has increased since 1981 (21,44). Nonetheless, loss of centralized control of screening criteria, quality control, and data analysis would be expected to slow progress in both identification of at-risk children and the means for assessing trends in poisoning risk.

In addition, a detailed study of data from Newark, NJ, for a 9-year period showed that the rate of positive toxicity cases in young children increased about 4-fold after funding for lead screening and public education programs was reduced (45). While this example was not linked to the change in screening support in 1981, it does show that those areas that decrease the efficiency of their lead screening services can expect to experience increases in the number of children with lead poisoning. Lead screening programs, when supported at a level that allows comprehensive screening and follow-up, are particularly cost effective. This can be demonstrated by comparing the costs of treating children who are hospitalized because of lack of early detection of exposure in one community with the costs of community screening programs in another.

According to O'Hara (46), the cost of repeat admissions to Baltimore hospitals for 19 lead-poisoned children in

1979 was \$141,750, or at least \$300,000 in 1986 dollars (~ \$16,000 per child). By comparison, the city of St. Louis spent \$404,453 for its 1985-1986 program year (1), and this allowed testing of 12,308 children, of whom 1,356 or 11.02% tested positive for lead exposure using the relevant CDC toxicity risk classifications. Of these positives, 849 were classified as Class II, 445 as Class III, and 62 as Class IV, the most severe level of toxicity [see report to Congress (1), for detailed discussion of screening classifications]. This amounts to approximately \$300 per child with early toxicity, or, overall, a benefit:cost ratio of about 53:1. This ratio does not take into account such additional costs as those for managing severe toxic cases, for medical follow-up care and treatment, remedial education, reduced lifetime earnings, reduced tax payments, or reduced earnings. Overall, monetized costs of the sequelae in lead toxicity cases are spelled out by U.S. EPA (24,37). In March 1987, the Committee on Environmental Hazards, American Academy of Pediatrics (47), issued a statement on childhood lead poisoning, including a recommendation that all children in the United States at risk of exposure to lead be screened for lead absorption at approximately 12 months of age with follow-up testing of children judged to be at high risk for lead exposure.

Currently, the Lead Contamination Control Act of 1988 (36), described earlier, contains provisions which call for the U.S. CDC to resume oversight and support of U.S. lead screening programs. The success of this legislative initiative will hinge on the actual level of funding.

**Environmental Hazard Identification and Abatement for Severe Poisoning Cases.** HAZARD IDENTIFICATION. When cases of toxicity were identified, mass screening programs for lead poisoning routinely made efforts to identify the sources of exposure as part of secondary prevention. In high-risk populations in urban areas, leaded paint was most commonly identified as the likely source of exposure via ingestion (20-22). However, other sources were implicated in other cases, since a significant number of toxicity cases were not identified with leaded paint.

**LEAD ABATEMENT METHODS.** As noted earlier, source-specific lead abatement actions as part of primary exposure prevention have had mixed success. For secondary prevention methods, the principal lead source at issue is leaded paint.

A careful examination of the information on reducing lead exposure by completely or partially removing leaded paint clearly shows that conventional methods often result in incomplete removal and often carry associated exposure risks.

In a prospective study, Chisolm et al. (48) observed that when lead-poisoned children are returned to "lead abated" structures, their PbB levels invariably increase to unacceptable levels. This does not appear to be a case of PbB increase from the endogenous release of bone lead, since children similarly exposed before treatment do not have such PbB elevations when placed in lead-paint-free housing.

Information has accumulated to show that leaded paint removal is hazardous to the workers doing the removal, and lead continues to be hazardous to occupants because

residual material has been removed to other areas accessible to young children. A major difficulty is the relatively high mobility of old, powdering (chalking) lead paint, which enters cracks and crevices, settles on contact surfaces, and readily sticks to children's hands. As Charney et al. (26) noted, abatement response to the paint dust problem may well be as important as removing the paint film.

The problem of continued exposure risk, either during or after leaded paint abatement, can be illustrated in the recent study by Rey-Alvarez and Menke-Hargrove (49). Rey-Alvarez and Menke-Hargrove examined lead-poisoned children ( $n = 13$ ) whose exposure had been exacerbated in varying ways when leaded paint was being or had been removed. Elevated PbB levels were further increased when paint was removed. Farfel and Chisolm (50) also document that traditional paint removal increases household dust and child PbB levels. These data augment the experiences of other investigators and make it clear that additional lead exposure during and after paint lead removal can occur.

Chisolm (51) has drawn attention to the need for some new approaches to the problem of removing lead from occupied housing. As noted earlier, the U.S. HUD is examining the relative effectiveness of paint lead removal strategies in its abatement demonstration program.

The above discussion deals with the elements of an ideally thorough lead removal approach as part of general risk assessment for paint lead. The extent to which even partial leaded paint abatement will lower PbB levels overall and would also be of value is an important matter but is more an issue for risk management (26).

A hidden assumption underlies efforts to remove leaded paint from the homes of children found to have lead poisoning: that the child will remain in the lead-abated home. In reality, there is high residential mobility among poor, inner-city residents. The long-term effectiveness of unsystematic "spot" abatement is questionable, perhaps even for the individual children for whom the effort has been made.

## Environmental/Biological Prevention Measures

This approach is analogous to that of primary prevention which combines nutrition and environmental control. Nutrition-based measures in the case of secondary exposure, however, might be more problematic. Specifically, one would be dealing here with children already showing signs of lead toxicity. A secondary nutritional approach would also require that the affected family take a more active role in exposure prevention, and this raises the issues of compliance, family budgets for adequate diets, etc.

## Extra-Environmental Prevention Measures

These measures essentially consist of legal sanctions to force the removal of lead from residences with documented poisoning cases. Such sanctions can be useful tools for responding to demonstrated and significant health risks. How effectively can one use a legal framework to expedite the rapid and safe removal of lead hazards from children's

daily environment? Conversely, can one conclude from available information that a real handicap for such action is the absence of supporting legal tools?

Answers to these questions in the available information are not easily found, but it is useful to examine a typical major screening program with a legal component and assess its contribution to overall abatement. In its summary of screening activities submitted to ATSDR (1), the City of St. Louis summarized its legal actions involving landlords and others who own housing or public-use facilities where lead poisoning had been found. A summary of 1985 court activity for leaded paint ordinance violations indicated a case load of 1,086, with 387 of the cases carried over from 1984. From this cumulative docket, 154 defendants were fined \$2,447, an average of \$16. Minor fines appeared to be the only measure at the city's disposal. The 1984 count was virtually identical to that of 1985, and the average fine for 1984 was the same as for 1985.

One cannot say whether minor fines as legal sanctions influenced the city's lead toxicity rate as identified from screening. In the most recent data, this rate was 11%—a rate that has remained about the same since 1978, owing to a number of factors. This case study does suggest, however, that a persisting high lead toxicity rate has not led to more effective legal measures.

## Summary and Conclusions

Both primary and secondary lead abatement methods have been examined in detail in this article and the report to Congress (1) which gave rise to it. Certain primary lead exposure prevention measures, i.e., the phasedown of lead in gasoline, the promulgation of ambient air lead standards for stationary source emissions, and phase-out of lead-soldered food cans have been effective in reducing overall childhood lead exposure.

By contrast, other lead sources and pathways, i.e., leaded paint in older U.S. housing and public buildings and lead in dusts and soils, remain as significant contributors to U.S. childhood lead exposure and intoxication. To date, little in the way of nationwide abatement efforts have been implemented for these routes and those that have been attempted have generally failed.

Finally, lead-contaminated drinking water in homes, schools, day-care centers, and elsewhere is currently a significant source of exposure for pregnant women and pre-school children. While there are proposed or newly mandated tapwater lead control measures, it will take time to evaluate their relative effectiveness.

As might be expected, the relative effectiveness of source-specific abatement actions in the United States has been closely linked to their implementation through centralized control strategies. Such measures using a centralized mechanism are typified by leaded gasoline phasedown, ambient air lead reduction, and regulation of lead in food sources.

Lead exposure routes that are widely dispersed throughout environmental compartments and that have impacts on diverse risk populations are inherently more difficult and costly to control. Such difficulties are manifested in a) the high levels of lead in the paint of over 21 million old housing

units and public facilities; *b*) lead-contaminated dusts and soils in urban areas and other sites affected by airborne and other lead deposition; and *c*) the millions of U.S. residential units and public facilities having contaminated tapwater due to lead-soldered plumbing, lead water line connectors, and lead-containing faucets or other fixtures.

An added problem in the case of either leaded paint or lead-contaminated dusts and soils is the extent to which meaningful lead reduction or complete removal can be achieved. This arises from the pervasive distribution of the contaminant within a dwelling unit or larger area, as well as the propensity for lead to be highly mobile. For example, it is quite difficult to maintain freedom from lead contamination at an abated site if neighboring sources provide recontamination by leaded paint weathering from exterior surfaces (29-32) or leaded dust reentrainment/redeposition, for example.

Given the limitations of piecemeal lead abatement actions, exposure reduction approaches are needed which are comprehensive and applicable on a neighborhood or other tract basis. This is not to say that individual sites of exposure, associated with identified lead intoxication, would not benefit from abatement efforts. The relative value of secondary versus primary exposure prevention methods is markedly affected by trends in the human toxicology and environmental epidemiology associated with lead exposure.

As the levels of lead exposure and associated levels of lead body burden, i.e., PbB considered safe, continue to be adjusted downward (22,47), it is increasingly clear that primary exposure control, rather than secondary methods such as screening plus medical intervention, will be the principal regulatory option. For example, PbB levels below 25 µg/dL are still of significant public health concern and are typical of exposure for millions of children and other risk subjects, but it is neither medically appropriate nor feasible to employ chelation therapy and other medical treatments in response to such body lead burdens. The only solution is reduction or removal of the sources of lead exposure.

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